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ON THE ÆTIOLOGY OF
PULMONARY PHTHISIS.*

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It is with some diffidence that I appear before an association of this character with a paper upon so hackneyed and, as some may think, so sterile a subject as the ætiology of phthisis pulmonalis; and more particularly so as I have no new facts to offer.

I am not even prepared to discuss, on the basis of my own knowledge, the latest and most important discovery in the pathology of this disease, viz., that of the *Bacillus tuberculosis*. The discovery, by Koch, of what appeared to be the specific germ of tubercular disease has put the pathology of the subject upon a new basis, and completely changed the drift of discussion. It has, it seems to me, so completely absorbed the attention of the medical world, that other and important matters are in danger of being entirely lost sight of. Yet, there are many important factors

* Read before the American Climatological Association, May 3, 1884.

in the pathology of phthisis besides the *Bacillus tuberculosis*.

Granting, as it appears we must, that the immediate exciting cause of miliary tubercles is the invasion of the body by these organisms, we have still to consider what are the conditions which precede this invasion, and render the tissues susceptible to it. The importance of examining carefully into the question of predisposition is evident, inasmuch as the seed can not ripen and bring forth its harvest of death, unless it falls upon a soil favorable to its development.

On the other hand, the exciting cause of tuberculosis—being ever present and unavoidable, except, perhaps, in some few favored localities—has, as yet, mainly a scientific interest; though we may hope that, in the future, we may be able to oppose its inroads more successfully than at present.

In studying the conditions which may be antecedent to, and prepare the way for, the development of phthisis pulmonalis, it will be convenient to adopt some form of classification. They may be arranged as follows;

1. Those conditions which are found in the bodies of ordinary healthy people.

2. Abnormities of anatomical structure. These are either the result of direct inheritance, or proceed from disturbance of health in the parents.

3. Conditions arising from imperfect development of the body after birth.

4. Acquired diseases in the lungs, leading to the formation of caseous matter or collections of decomposing pus.

5. Such conditions of life as favor the occurrence of struma and caseous pneumonias.

It may be allowed, though there is as yet no proof of it, that, by direct inoculation, a *healthy* human being may be rendered tuberculous. But I think it fair to assume that,

in the vast majority of cases, some or many of the predisposing causes mentioned are in existence prior to the advent of active tuberculization.

I. The conditions which are found in the bodies of ordinary healthy individuals, and which favor, indirectly, of course, the occurrence of this disease, exist in the structure of the lungs and thoracic walls.

In studying the anatomy of the lung, we find that the respiratory mucous membrane undergoes important modifications, as it passes from the bronchial tubes into the bronchioles and alveolar structures. When the caliber of the tubes is reduced to a certain diameter, the mucous glands disappear, and the tenuous walls are lined by a single layer of epithelium.

In the alveolar passages, infundibula, and vesicles, the epithelium, which was columnar in form during foetal life, becomes flattened when respiratory dilatation is established, and forms a single layer of flat cells, lying directly upon the vessels and connective tissue of the alveolar septa, with no submucous layer, and no mucous glands. It is true that epithelial cells have, everywhere, a glandular action, and separate something from the blood. But, in this case, the activity of the cells is in the direction of the excretion of gases, aqueous vapor, and, probably, of certain effete organic substances, from the blood in the pulmonary capillaries. It can not be said that mucus is secreted, below the line at which the mucous glands cease to exist.

The physiology of respiration teaches us that the air contained in the bronchioles and alveoli is never bodily changed, except when collapse and subsequent rehabilitation occur. In other words, these small cavities never contract sufficiently to entirely expel their contents. Nor can any act of coughing, or other effort, cause them to do this; so that, if any substances accumulate in them, they must either

be liquefied and reabsorbed, or, if some moisture exists to favor movement, they may be slowly expelled piecemeal; or, should neither of these occur, they will remain, and either undergo some form of decomposition, usually the caseous metamorphosis, or organize into a new tissue. The absence of mucous glands to supply moisture, and the obstacles to the expulsion of dry substances from the alveoli, thus appear to be two very important conditions, resident in the normal structure of the lung, and indirectly favoring the establishment of caseous foci.

Besides these, there are certain circumstances affecting both the sanguineous and aerial circulation in the *apices*, and favoring the location of disease there.

The thorax is so constructed that the muscles of *inspiration*, acting upon both sides simultaneously, reduce the internal pressure upon all sides; for, while the muscles do not act directly upon the extreme apices, where the pleuræ project through the superior opening of the thorax, nor upon the mediastinal surfaces, still, the cavity being a closed one, any change in pressure, not too rapidly induced, will be felt equally in all its parts.* In this way, the apices and mediastinal regions will expand, equally with the other portions of the lung, and the air will be invited into them.

During *expiration*, however, the conditions are different. The thoracic walls, which have been expanded by the traction of the inspiratory muscles, return to the expiratory position through the agency of three factors—viz., the elastic contractility of the lungs, the resilience of the chest-walls, and the upward pressure of the abdominal contents. It will be seen that only one of these forces—viz., the elasticity of the lungs—acts upon the extreme apex. In this locality, the cavity is closed in by a strong fascia, attached

* For a careful study of the subject of intra-thoracic pressure, see Dr. Garland's work on "Pneumono-Dynamics."

to the scaleni and first rib. During *inspiration*, this fascia, if moved at all, must descend. In forcible inspiration, its *relative* descent is well marked.* During *expiration*, it either remains stationary, or ascends. With a forcible expiration, as in coughing, its ascent is considerable. It results from this that, while the other portions of the lung have three forces acting, to favor the expulsion of air from the alveoli, in the apices there is no other force than the contractility of the lung itself, and this is somewhat counteracted by the ascent, or tendency to ascend, of the cervical fascia.

The ordinary conditions, then, are not favorable to the free ventilation of the apex.

The pulmonary circulation can not be perfectly performed, save in the presence of free movements of expansion and contraction, and the circulation in the apex, where these are diminished, is, consequently, not as free as in the other portions of the lung. In addition to this, the force of gravity tends to diminish the amount of fluid in the apex. It is the constant observation of pathological anatomists, that the apices are the dry and anæmic portions of the lungs. They are the illy-nourished portions. If, now, owing to an inherited peculiarity of structure, to vices of general nutrition, or to old age, the movements of the upper portion of the chest are still further restricted, or, what is more important, if the contractility of the lung is diminished, both air and blood will have but a feeble circulation, and, should any other cause, as a chill, intervene, to determine the flow of blood to the lungs, the vascular paresis will be felt most at the weak apices, and the tendency to effusion will be the greatest there. But the inflammation will not be a sthenic, croupous one, as it

* See Dr. W. S. Forbes, The Diaphragm, etc., "Am. Jour. of the Med. Sci.," July, 1880.

is when the hyperæmia is more active. Venous engorgement will be in the ascendant, the hepatization slow in appearing and disappearing, and the infiltration and cell-accumulation in the connective tissue predominant.

Another element of importance in the effect of the normal structure of the body upon the tendency to phthisis, is the effect of the expiratory muscles upon the thorax in the act of coughing. This can only be operative in cases in which disease has already started in the apex.

The muscles of expiration act mainly upon the lower portion of the chest.* In the sudden and violent expiratory efforts of coughing, the lower and middle portions of the lungs are, as it were, violently compressed. The effect of this compression is, I believe, to force the air upward toward the trachea, with such power and in such volume that it can not find an immediate exit, but a portion of it will be driven into the bronchi of the upper lobe, in which the current will be reversed, the air returning toward the alveoli. Should the cause of the cough be an ordinary bronchitis, the result will be an emphysematous dilatation of the alveoli of the upper lobes, either temporary or permanent, according to the duration, etc., of the bronchitis.† If the cough results from a catarrhal process at the apex, it will be readily seen that this reversal of the current of air will tend to force the morbid products back into the air-sacs, and favor their retention and caseation.‡ While fluids

* *Vide* "A Lecture on Cough, as a Symptom of Disease," delivered before the Chi Phi Delta Fraternity, and published in the "American Journal of Neurology and Psychiatry," July, 1884.

† The author does not mean to be understood as affirming that this is the sole cause of emphysema.

‡ Professor D. J. Hamilton, of Aberdeen, in his work on "The Pathology of Bronchitis," etc. (London, Macmillan & Co., 1883, p. 156 *et seq.*), lays stress upon this dryness of the apex, particularly as favoring the caseous metamorphosis. He thinks it due, not alone to the gravita-

gravitate from the apex toward the base, dry residues and collections of cells, always difficult of expulsion from any portion of the alveolar structure, are retained here with especial tenacity. The influence of these normal peculiarities of structure in favoring the location of phthysical processes in the extreme apices of the lungs, is of immense importance, and should never be lost sight of in discussing the pathology of consumption.

II. Abnormalities of anatomical structure are of very great ætiological importance, as to them is due the constitutional predisposition of those unfortunate persons who, by direct inheritance of similar peculiarities from their parents, or as a result of the operation of impaired conditions of life upon the latter, have come into the world with a tendency to pulmonary phthisis. Besides *anatomical*, I might have mentioned *physiological* peculiarities; but, without lingering over the question of the interdependence of structure and function, we may assume that the two go together, and that mention of the one implies the presence of the other.

The most evident deviations from the normal standard are to be noted in the general structure and external appearance of the body. Certain subjects present the well-known appearance of the phthysical habit. Aretæus is quoted by Laennec * as drawing the following picture: "Extreme whiteness of the skin, bright-red color of the malar eminences, narrowness of the chest, whence arises the wing-

tion of fluids toward the base, but also to the increased pressure in the alveoli during the act of coughing. This increased pressure, he believes, squeezes the liquids through the alveolar walls into the connective tissue, whence they are removed by the "absorbents." Whether the absorption from the air-passages does not rather occur during inspiration, when the pressure is removed from the lymph lacunæ, is a question into the discussion of which I will not now enter.

* Laennec, "Treatise on Mediate Auscultation," etc., p. 312.

like form of the scapulæ, and the slenderness of the limbs and of the body, although such individuals have a certain degree of adipose and *embonpoint*."

This, as Laennec justly observes, indicates, particularly, the appearance of the hæmoptysical. The long thorax, with sharp epigastric angle* and small muscles, is known to all clinical observers as of bad omen in regard to the impending development of phthisis. So also are the subjects of scrofulous habit recognizable by signs which it is not necessary to enumerate here. All these appearances indicate abnormalities of anatomical structure, favoring, more or less directly, the occurrence of phthisis. But, of late years, a new field of anatomical study has been opened by Professor Beneke, of Marburg.† This is no less than the collection of data for the determination of the question, whether the peculiarities and anomalies of constitution are not dependent upon differences in size, weight, etc., of the internal organs. Already, the study of anthropometry had, in the hands of Quetelet, Roberts, Bowditch, and others, given interesting and valuable results, so far as the external anatomy was concerned; but Beneke and Thoma,‡ taking their hint, perhaps, from Virchow's observations on the arterial system in chlorosis, have made extensive series of measurements and weighings, in order to establish the anatomical basis of individual differences and anomalies. As Beneke observes: "We can not comprehend the working

* By the term *epigastric angle*, I mean to designate the angle formed by the convergence of the inferior borders of the costal cartilages. It is bisected by the ensiform appendix.

† F. W. Beneke, "Die anatomischen Grundlagen der Constitutionsanomalien," Marburg, 1876; "Constitution u. constitutionelles Kranksein des Menschen," Marburg, 1881.

‡ R. Thoma, "Untersuchungen über die Grösse und das Gewicht der anatomischen Bestandtheile des menschlichen Körpers im gesunden u. im kranken Zustande," Leipzig, 1882.

of the human machine as a whole, unless we possess a knowledge of the absolute and relative proportions of all the separate parts of it. . . . There can be no doubt that an organism with a large heart, capacious arteries, small lungs, and large liver, will work out an entirely different result from one of the same age with small heart, narrow arteries, large lungs, and small liver." The result of his observations in regard to phthisis, up to 1881, appears to be that the ordinary phthisical subjects, with caseous pneumonias, are distinguished by the possession of relatively small hearts, narrow arteries, large lungs, small livers, and *short alimentary canals*.

Thoma's observations, which have been conducted on a very extensive scale, and are concerned to a greater extent with the establishment of averages for normal structure, on the calculus of probabilities, show, in the field of pathology, a somewhat similar result.

The labors of these anatomists, while still too limited to give us accurate results, point, undoubtedly, to an anatomical structure, either directly transmitted, or else inherited, as a result of unfavorable conditions of life on the part of the parents, underlying, and standing in a causal relation to, the phenomena of special constitutional diseases.

III. The conditions arising from imperfect development of the body after birth, and which render the subject vulnerable to the tubercular infection, or favor the development of the caseation which immediately precedes it, are numerous and difficult of classification. They are, in the first place, a state of low vitality. By this, I mean a state in which the volume of blood is diminished, the body dwarfed in one way or another, the chylopoëtic system feeble in its functions, and the nervous system ill nourished and inadequate to the ordinary duties of life. The circulation and respiratory movements are weak, the intellect, though sometimes

bright, of a low order, and the capacity for work limited. The lungs are poorly nourished, poorly ventilated, and liable to the catarrhal and interstitial forms of inflammation.

Again, we meet with the serofulous diathesis. This, though often inherited, is frequently acquired, as a result of exposure to various devitalizing influences. I will not take up the time of the Association with a discussion of the pathology of this protean disease, but simply allude to it as one of the dyscrasie which favor the formation of cheesy deposits, and the diminished vital activity which invites the invasion of parasitic diseases.

The exanthematous fevers seem to predispose to phthisis, through their tendency to inaugurate the strumous diathesis. Typhoid fever presents some curious analogies to tuberculosis, or else some cases have been confounded by those who have observed them. An interesting collection of such cases has been made by Dr. Creighton in his work on "Bovine Tuberculosis in Man." They are, at least, a most interesting pathological study.

During youth, and in adult life, numerous circumstances occur which either directly deprive the body of sufficient nourishment, cause an excess of consumption over alimentation, or induce diseases which interfere with primary or secondary assimilation. These circumstances, sometimes obscure, sometimes impossible of detection, lead to a condition of inanition or wasting of the body which does not, I believe, render it specifically liable to *tuberculosis*, but favors the occurrence of adynamic catarrhs, asthenic pneumonias, and other chronic inflammations. This wasting of the body, precedent to the active manifestations of phthisis, has attracted the notice of pathologists and clinical observers from the earliest times, and, owing to the obscurity of its origin, has been considered a part or stage of pulmonary consumption. There has been much speculation as to the

nature of this profound disturbance of nutrition, and many are the theories in regard to it which have vexed the medical mind. Dr. Horace Dobell, in his work on "The True First Stage of Consumption," in 1867, and "On Loss of Weight, Blood-spitting, and Lung Disease," in 1879, has given this subject a most thorough and able discussion, bringing to bear upon the question many original ideas and observations. Dr. Dobell thinks that this wasting or inanition of the body, prior to the development of pulmonary phthisis, should be considered the first stage of the disease, though he admits the specific nature of the tubercular virus. It is, undoubtedly, the first link in the long chain of morbid physiological actions, which has its beginning in a state of almost perfect health, and its end in the total disintegration of the body; and the subject can not be properly discussed without admitting this as a very important element in many cases. On the other hand, in many cases, the signs of pulmonary disease appear before any perceptible loss of weight is observed.

The idea of Dobell and others, that pancreatic inefficiency is at the bottom of the mischief, is one which we can scarcely accept. The pancreas is an organ with the pathology of which, beyond the occurrence of certain inflammations and tumors, we are not well acquainted. My own impression is that, like the other salivary glands, its diseases are few and simple. We know, however, that in debilitated states, as anemia, nervous depression, and imperfect oxygenation, the digestive fluids are not normal in quantity or quality. There is a condition of atonic dyspepsia. The food substances introduced into the stomach and not properly digested, and undergo decomposition, with the formation of fatty and other organic acids and products of the disintegration of albuminoid compounds. These irritant bodies, by their action upon the digestive

mucous membrane, engender a catarrh, which still further interferes with the primary assimilation. In some instances where, as far as we can see, loss of weight continues, in spite of the fact that the patient takes the usual quantity of food and digests it, there is, probably, a fault in the original construction of the body, such as is spoken of by Professor Beneke as existing in the bodies of the victims of caseous pneumonic phthisis, viz.: "A relatively small heart, a relatively narrow arterial vascular system, relatively large lungs, a relatively small liver, a relatively short, small intestine."* This lack of equilibrium between the nutritive and respiratory systems would seem, *a priori*, sufficient to account for much of the wasting that is observed in these persons.

The statement of Rokitansky, quoted by Dr. Dobell, and, as the latter says, confirmed by Dr. C. Meymott Tidy,† that the blood in phthisis is in a state of hyperoxidation, is worthy of further investigation.

The further circumstance that phthisical patients, in spite of the pulmonary implication, are not usually cyanotic, is of cognate importance. But, in whatever way the details of this pathology of nutrition may be settled, Dr. Dobell has certainly done a great service to the profession and to humanity, by his recognition of the importance of the digestive inefficiency in phthisical persons, and by devising means for remedying the defect.

The importance of looking after the primary assimilation, in those who are suffering from inanition, either with or without phthisical complication, has been very strikingly illustrated by the experiments of Dr. Debove, and others, in super- and artificial alimentation.‡ The

* "Constitution u. constitutionelles Kranksein," p. 47.

† Dobell, "Loss of Weight, Blood-spitting," etc., p. 148.

‡ A very interesting account of Dr. Debove's methods and results may be found in the "N. Y. Medical Journal," April 19, 1884, p. 429, by Dr. H. B. Millard.

results of Debove, whose main reliance is on pulverized lean meat, compared with those of Dobell and others, if they prove anything, show that it is not a lack of any one alimentary principle that needs to be supplied in these cases, but that the need is for very easily digestible and assimilable foods, irrespective of the exact proportion of hydrocarbonaceous, albuminous, or other ingredients. The advocates of cod-liver oil and fats have, in their enthusiasm, overlooked the fact that the wasting in inanition, and particularly in phthisis, involves the muscles as well as the adipose tissue. It involves almost everything, the wasting process, however, showing itself variously in different organs. The liver, notably, shows its lack of nutrition in a molecular disintegration of its cells, with the local production of fat.

The importance of this defective nutrition, as a predisposing cause of phthisis, can not be exaggerated. Though recognized long ago, and insisted upon, particularly by Dr. J. H. Bennet,* it is receiving more attention now than formerly, and better practical results have been obtained, since our knowledge of the physiology of digestion has been turned to account in its treatment.

It will not be out of place, to refer, particularly, to the general constitutional weakness that results from long confinement in-doors. Whether this depression is a result of the continual action of the "gloomy passions," as was thought by Laennec† to be the case with those who spend their lives in conventual seclusion; or whether it is not, rather, induced by lack of sunlight, ventilation, and the varying sense impressions of ordinary life, needs little discussion. That the mortality from phthisis among cloistered

* J. H. Bennet, Reynolds's "System of Medicine," vol. iii, p. 546, 1871: "On Pulmonary Consumption."

† *Op. cit.*, p. 310.

orders is very high is, I believe, true. These instances are only of scientific interest, inasmuch as prophylaxis is impossible.

IV. Acquired disease in the lungs, leading to the formation of caseous matter, or collections of decomposing pus, is, of all causes, the most important in the ætiology of phthisis pulmonalis. This statement will immediately raise the question whether I am not confusing the disease itself with a cause of the disease; and, further, admitting the anatomical distinction between caseous pneumonias and miliary tuberculosis, are they not but different forms of the same disease? There has been so much theorizing and hair-splitting in the discussion of these questions that it is with great timidity that I approach it. But, if the Association will bear with me for a few moments, I will endeavor to keep out of the deepest part of the mire, and wade through as rapidly as possible.

In the first place, I think that we should make a clear distinction between the simple occurrence of a pulmonary inflammation, leading to localized cheesy metamorphosis, and the general collection of morbid conditions which go to make up a case of what we all, immediately, recognize as pulmonary consumption. One is a local process, more or less extensive, which may become latent, and never give rise to further trouble, or may even undergo resolution; the other is a constitutional dyscrasia, involving the most profound nutritive changes in the body at large, and can seldom give a better result than by having its course somewhat prolonged. I would not by this be understood as holding the doctrine that phthisis pulmonalis is a specific disease, analogous to the zymoses. There is, to my mind, no evidence of its specificity, prior to the development of miliary tuberculosis. It may be proved hereafter that caseous metamorphosis is the result of specific

infection, but I think it is not proved as yet. The doctrine of Virchow and Niemeyer, that caseous pneumonia and miliary tuberculosis are two different things, is not, so far as I can judge, overturned by the immortal discovery of Koch.

What, then, is phthisis pulmonalis? *Phthisis pulmonalis is a chronic, wasting disease, of varying ætiology and pathology, whose essential anatomical lesion is the result of a destructive process in the lungs, and whose most prominent symptoms are emaciation, cough, hæmoptysis, fever, and colliquative discharges.* The important points in the ætiology are, first, conditions which affect the general metabolism of the body in such a way as to give a weak nutritive power, and to disturb the proper equilibrium between the physiological actions of its different organs; second, inflammations of the pulmonary texture, which lead to its destruction. In order to make my meaning clear, I may refer to our ordinary colloquial expressions. If we examine a patient who is emaciated, coughs, and perhaps has had hæmoptysis, but find no evidence of disease in the apices, we say "he is on the verge of phthisis"; he may recover, but he will probably go into consumption. What we mean is, that the clinical picture is not yet complete, that one or more of the essential conditions are lacking, and the distinction is recognized as such an important one that we base our prognosis upon it. Another patient may give evidence of slight disease at the apex, but without marked emaciation, or without fever, and we say the same thing—"He is in great danger of having phthisis." Such a patient may never develop the rest of the lesions. He may recover. Of course, some signs are more highly diagnostic than others—*e. g.*, hæmoptysis is more so than cough; night-sweats more so than emaciation; a peculiar appearance of the larynx more so than bronchitis. The apical catarrh, and fever, are the most important of all;

but still, they are only a part of the disease. And in this, as in other diseases, there may be great individual variations. This has undoubtedly been the cause of much of the confusion of thought that has existed in regard to the true nature of phthisis pulmonalis. It originated when diseases were regarded as entities, and before the advent of modern pathological physiology.

Tuberculosis is, also, a part of the vast majority of cases of phthisis, but is, generally, one of the late occurrences, hastening the fatal termination. In some cases, it is an early phenomenon, and may, indeed, play the leading *rôle* in the morbid processes in the lung. It can not, however, in the great majority of cases, be called a cause of phthisis, inasmuch as it does not appear till after the disease is well established. Acute tuberculosis should be classed by itself, among the specific infectious diseases.

Before leaving this part of my subject, I wish to say that, in these remarks upon the definition and pathological character of phthisis pulmonalis, I have simply presented what seems to me to be the state of the case at present. I am not advocating any pet theory, as I have none.

The inflammations referred to as leading to caseation and the accumulation of decomposing pus are chronic bronchitis and peribronchitis, resulting in bronchiectasis; chronic catarrhal or desquamative pneumonia; and acute pneumonia, in which the products of the inflammatory stage are of such a character, or so located, that reabsorption and expectoration can not occur. It is affirmed by some, particularly by Dr. Leaming, of New York, that pleuritic inflammations are, by extension into the connective tissue of the lung, a frequent indirect cause of caseous metamorphosis. While I believe that the doctor overestimates the frequency of this occurrence, I am far from denying its possibility. It may be possible, also, though I know of no evidence bearing

directly upon the question, that the retention and decomposition of pus in the ventricles of the larynx may afford a supply of irritative material which, being inhaled into the deeper passages, can set up chronic bronchitis, peribronchitis, and pneumonic processes.

It is not within the scope of this paper to discuss the pathological histology of the lesions referred to above, but a few observations may be allowed in explanation of the statements made. In regard to the chronic bronchitides and pneumonias further remark is unnecessary. The acute pneumonias, however, require a little further discussion, as there has been much contention in regard to their ætiological relations.

That acute pneumonia does occasionally terminate in phthisis pulmonalis will be granted by every clinical observer. When, however, we inquire whether this sequel is only observed in cases of catarrhal pneumonia, or whether it does not also follow the croupous form, we meet with conflicting views. The difficulty lies in distinguishing, ante mortem, between croupous and catarrhal pneumonia. Cases of catarrhal or desquamative pneumonia are occasionally met with which can not be diagnosticated except by their subsequent history, or by post-mortem investigation. If any one should maintain, as Buhl* does, that the cases which terminate in this manner are all cases of desquamative pneumonia, it would be very hard to disprove it. The immense pathological and clinical experience of this observer entitles his opinions to the very greatest respect. And, indeed, it would appear, *a priori*, that the great majority of cases in which acute pneumonia terminates in phthisis are really instances of catarrhal, and not of croupous, inflammation. If we bear in mind that the catarrhal

* Buhl, "Mittheilungen aus dem pathologischen Institute zu München," 1878,

pneumonias are the adynamic pneumonias; that a more or less cachectic condition is necessary to the occurrence of caseation, at least to any extent; that the increased proliferation of epithelium and collection of cells in the alveolar septa are the conditions necessary to caseation; and that these do not occur in croupous pneumonia—we have already good reason for doubting the liability of the latter disease to eventuate in the lesions of phthisis. But when we remember the additional fact that post-mortem examinations show that croupous pneumonia occurs mainly in robust people, or in those devoid of any evidences of a cachectic state, while the catarrhal or desquamative type shows itself in those who are of weak and infirm constitution, the strength of Buhl's position appears still greater. Owing to the essential differences between the histology of phthisical lungs and of those affected by the croupous pneumonic process, it must be admitted that, supposing that a croupous pneumonia eventuates in a phthisical lesion, it must, in the transition, have altered its characters and assumed those of the desquamative form.

The practical lesson to be derived from these observations is that, inasmuch as it is not possible in all cases, during their early stages, to diagnosticate croupous from desquamative pneumonia, the latter stages of this disease should always be most carefully watched, with a view to counteracting any tendency to chronicity. It is especially important to repeat the physical exploration of the chest from time to time, so as to keep track of what is going on in the lung; for, in cases of chronic pneumonia, the general appearance of the patient will sometimes improve, and he may express himself as feeling quite well, when an examination of the chest shows that solidification of the lung is still present. I have seen patients who, several weeks after the beginning of a pneumonia, had marked dullness upon percussion, with

bronchial breathing and bronchophony over the affected area, although they expressed themselves as feeling as well as ever, and, to superficial observation, appeared so.

V. This study of the predisposing causes of phthisis would be incomplete, without some more particular reference to those conditions of life which have been alluded to as favoring the occurrence of struma and caseous pneumonias. They are the conditions which it is one of the main objects of this Association to investigate. If the true relationship of phthisis to serofula were more clearly ascertained, an important advance would have been made in the study of our subject. It seems, however, that the conditions which favor serofulosis also favor the development of phthisis. In the present state of geographical pathology, it is difficult to make any positive statements, but we may say that, as a general rule, the two diseases go hand in hand. But to this there are some notable exceptions. For instance, in Sweden and Norway, where phthisis is rare, serofula is said to be exceedingly common,* though in Iceland and the Faroe Islands the former † is almost unknown and the latter rare. In the elevated regions of South America, again, where phthisis is very uncommon, serofula is said to prevail extensively.‡ The meaning of these facts, if they are facts, is not apparent. We should bear in mind that the observations collected by writers are made by men of varying qualifications; that some have different ideas of pathology from those entertained by others; and that, in the absence of post-mortem records, made by reliable anatomists, much confusion may arise. It should also be remembered that many of the recorded observations are made by travellers, whose only or

* A. Hirsch, "Handbuch der historisch-geographische Pathologie," Stuttgart, 1883, 2te Abth., s. 433.

† Lombard, "Traité de climatologie médicale," t. ii, p. 49.

‡ A. Hirsch, *op. cit.*, Abth. ii, s. 438.

chief sources of information are the accounts obtained from those with whom they come in contact. It is true, however, that many of the conditions which would favor the increase of scrofula also favor the development of phthisis. Residence in damp and gloomy places, where excess of moisture and lack of sunlight combine to interfere with the normal physiology of the body, is a prolific source of the disease. Bad food, particularly such as taxes the digestive power without furnishing a full proportion of the stimulating nitrogenous elements, particularly if combined with the former circumstances, should be considered as a formidable enemy to the proper nutrition of the body. A. Vogel * doubts the ability of these causes to produce the tuberculous diathesis, except in those who are constitutionally predisposed to it.

That overwork, particularly where it is carried on indoors and on a poor diet, is a very frequent cause of phthisis pulmonalis, will, I think, be admitted by any one who has had practical experience in our large cities. The difference in health between domestics, who are actively employed in well-appointed households, and factory girls, confined all day in one position, almost, in close, dusty rooms, is so noticeable as to attract the attention of the most careless observer. This, however, may be partially due to other causes.

All kinds of dissipation may be reckoned as causes of this disease, unless, it may be, excessive, continual drinking, by those who are not overworked and who are liberally fed. Such persons tend to excessive development of adipose tissue and blood, and to diseases of the abdominal viscera. If they die of pulmonary disease, it is usually of acute pneumonia.

A frequent cause, which is particularly observable in the present age, when great masses of population are

* "Diseases of Children," translated and edited by H. Raphael, M. D., New York, D. Appleton & Co., 1873, p. 539.

shifting their homes, and passing from one country to another, where they meet with entirely new conditions, is the change of vital operations caused by such a transition. It is a matter of common observation, in our large eastern cities at least, that large numbers of the emigrants, but more particularly the children of emigrants, fall victims to this terrible malady. In my own observation, they have been mainly the children of Irish parents, born upon American soil; but it may be true of other nationalities. The Irish emigrants are more prone to remain in New York and Brooklyn than those of other nationalities are, so that we have a better opportunity to watch them. I have been told by my friend Dr. Newton, of Rutland County, Vermont, that the same holds true among the Welsh quarrymen, who are employed in the slate and marble industries of that State. The explanation of this peculiar tendency in the children of emigrants is, probably, to be explained by a comparison of the conditions of nutrition and respiration existing in the parent country, with those prevalent here. An organism, whose structure is, by heredity, adjusted to the climatic and dietetic conditions of the British Isles, and, particularly, to the farinaceous and fatty food of the Irish peasantry, is not calculated to maintain its vital equilibrium under the altered circumstances by which it is invested in the New World. While the fully developed adult may succeed in accommodating himself to it, it is not so easy for an infant to develop healthily, under circumstances so entirely different from those under which its ancestors have lived for centuries.

These great ethnic changes were appreciated, years ago, by Dr. Daniel Drake, who even prophesied that some pathological phenomena would result from them.* Another

* Daniel Drake, M.D., "On the Principal Diseases of the Interior Valley of North America," Cincinnati, 1850, p. 648.

and equally important observation, if it is correct, is that the inhabitants of the Antilles and of the Polynesian Islands were free from phthisis before the advent of civilization among them. Though the evidence seems strong, we are almost inclined to doubt it; but, if it is true, it may indicate either one of two things. Since the specific infection of tuberculosis has been discovered, it may be asserted that the European settlers introduced the virus among the aborigines of the newly discovered countries. Or it may be said that the change in their mode of life has affected them, very much as emigration would. "The transition from a nomadic or savage life to civilization," says Lombard, "is one of the most favorable circumstances for the development of phthisis."*

In regard to the special influence of climate upon the tendency to phthisis, I will say but little, as others are to give it a more thorough treatment than is possible in such an essay as the present. What the essential element is which gives immunity, it is difficult to determine, except in a general way.

That elevation is very important there can be no doubt. It is generally admitted that elevated and dry regions are more or less exempt from the disease.

Equability is certainly favorable to the longevity of those already affected.

Temperature is of less importance.

But, while admitting this much, we must not forget that some low countries are free from it. It is said not to exist among the Kirghiz tribes, who inhabit a region which lies below the level of the sea. It is also not found in some countries the climate of which is very changeable, as Iceland, the Faroe Islands, and some of the Hebrides.

Why it is that moist localities in New England should

* Lombard, *op. cit.*, t. iv, p. 422.

be infested by it, while the islands referred to are almost, if not entirely, free from it, is worthy of investigation. Stress should be laid upon the fact that Iceland is of volcanic formation. Very slight vegetation is found upon it, and, in the valleys, where there is more or less swamp, the ground, even in summer, is frozen, a short distance below the surface. Then, the inhabitants live largely upon a diet of animal food.* It is essentially a marine climate, and that is not unfavorable in its influence upon phthisis.

It is stated by Laennec† that in France phthisis is far less frequent upon the coast than in the interior. Probably, the immunity of these people is due to the marine climate, absence of miasmatic emanations from the soil, and their animal diet. It would seem to throw some doubt upon the importance of moisture, simply as moisture, as an ætiological factor. This very short and imperfect reference to the climatic influences may serve one useful purpose. A careful scrutiny of the facts, presented and alluded to, will, it seems to me, lead us still farther toward the recognition of the importance of *errors of general nutrition* as ætiological factors in the history of pulmonary consumption.

As the fundamental sciences of anatomy and physiology, both normal and pathological, are more fully investigated, new light will be thrown upon this subject, and it is to be hoped that, at no distant day, our knowledge may lead to the solution of many of the problems which now baffle our most determined efforts.

* The herbivorous animals are most easily inoculated with tuberculosis.

† Laennec, *op. cit.*

pure
but not
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for
cattle

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EDITED BY
Frank P. Foster,
M. D.

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